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Monitoring of Evoked Potentials During Spinal Cord Ischaemia: Experimental Evaluation in a Rabbit Model

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Objectives: *somatosensory evoked potentials (SEPs), spinal evoked potentials (Spinal-EPs), and motor-evoked potentials (MEPs) were monitored in a rabbit model of spinal cord ischaemia to evaluate their accuracy and relationship to clinical status*

Methods: *a modified rabbit spinal cord ischaemia model of infrarenal aortic occlusion for 21 min was employed (30 rabbits) After baseline SEPs, Spinal-EPs, and MEPs were obtained, evoked potentials were recorded continuously during and after clamping of the aorta (30 min). Neurological outcome at 24 h was correlated with evoked potentials, and histopathological findings*

Results: *fifteen animals became paraplegic. MEPs were always abolished after clamping of the aorta while Spinal-EPs and SEPs remained. The sensory evoked potentials (SEPs and Spinal-EPs) were the least sensitive to spinal cord ischaemia, and their presence had no correlation with the final clinical status (50% of false negatives). This was consistent with histopathological examination that showed damage almost entirely confined to the anterior horn, while the dorsal columns were generally well preserved High spine MEPs evoked by twitch stimulation was the best predictor of clinical outcome (0% of false negatives, 0% of false positives).*

Conclusions: *SEPs and Spinal-EPs cannot be used as safe monitors of ischaemia of the spinal cord. High spine MEPs evoked by twitch stimulation was the most useful for real-time evaluation of spinal cord ischaemia, and the best predictor of neurologic outcome during reperfusion.*

Key Words. Paraplegia, Evoked potentials, Monitoring spinal cord function

Introduction

Despite the use of several adjunctive techniques and drugs, paraplegia continues to be the most dreaded complication following thoracoabdominal aneurysm resection. To detect deterioration of spinal cord function and to prevent permanent damage by reimplantation of intercostal vessels lying within the excluded segment, a number of intraoperative monitoring methods have been developed.[1,2] Current techniques of monitoring spinal cord function evaluate primarily the sensory pathways. Although monitoring somatosensory evoked potentials (SEPs) is considered by some investigators to be a sensitive means of early warning of impeding paraplegia, it has also been criticised as having many drawbacks.[3–6] SEPs fail to monitor directly the function of anterior spinal cord motor tracts, and the ischaemia of peripheral nerves as a result of clamping of the aorta interferes with the interpretation of the SEPs recordings.[7] Moreover, SEPs appear to be dependent on body temperature, anaesthetic drugs (especially halothane), haemodilution, hypoxia, and hypotension.[11] To correct some of the disadvantages, some authors[8,9] have suggested an alternative by stimulating and recording the spinal cord with electrodes inserted into the epidural space at different levels. This technique of spinal evoked potentials (Spinal-EPs) avoids the problem of peripheral nerve ischaemia associated with aortic clamping. Although some recent papers report a good clinical correlation,[10,11] the impact of these methods on prevention of paraplegia in the clinical setting has been limited by false positive and false negative results.[5,6,12–14] Because of this, others have suggested monitoring of motor evoked potentials (MEPs).[15–18] A variety of techniques have been developed to stimulate motor tracts, but each technique has undesirable features which limit its usefulness for routine intraoperative monitoring [19] However, Haan *et al.*[20] have recently demonstrated that monitoring myogenic motor responses to transcranial stimulation is clinically feasible and effective in detecting spinal cord ischaemia during operations for thoracoabdominal aneurysms, although careful planning

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of the anaesthetic technique is necessary. In this paper, we describe a method to evoke a muscle action potential after direct stimulation of the spinal cord through needle electrodes inserted into the spinous processes of vertebrae. This compound muscle action potential was assumed to be a response that was mediated through the motor pathway, since the myogenic responses are entirely specific for the status of the motor neurons in the anterior horn gray matter.[17,21]

The purposes of this study have been to test various monitoring methods (SEPs, Spinal-EPs, and MEPs) in a rabbit model of aortic occlusion to evaluate their accuracy and to determine their correlation with clinical status. Because the rabbit has a segmental arterial supply to the spinal cord, occlusion of the infrarenal aorta will result in spinal cord ischaemia, with minimal haemodynamic variation.[22] Furthermore, allowing the rabbit to recover permits clinical evaluation spinal cord ischaemia so that neurologic changes can be assessed. This method avoids the tremendous variability of collaterals to the spinal cord, and the marked haemodynamic and physiologic changes associated with thoracic aortic cross-clamping observed in other animal models, which can interfere with the electrophysiological assessment.

Material and Methods

Thirty New Zealand white rabbits of both sexes (2.0–3.5 kg) were used for this study. A modified rabbit spinal cord ischaemia model of infrarenal aortic occlusion for 21 min was employed. This study was approved by the Institutional Review Board, and all experiments were conducted in compliance with the "Principles of Laboratory Animals Care" formulated by the European Council and published by the National Institute of Health (Spain, Royal Decree 223/1988, B.O.E. No 67: 8509–12).

Surgical procedure

The animals were anaesthetised with intramuscular ketamine hydrochloride (40 mg/kg). Intraoperative fractional doses were used to maintain appropriate levels of anaesthesia and prevent the need for endotracheal intubation and mechanical ventilation. The animals were immobilised on the operating table, and the fur in the neck, lumbar and dorsal spine, hind limb, and abdomen was clipped with electric shears, and the skin was prepared with iodine solution. After local infiltration of mepivacaine hydrochloride (10 mg/kg), a midline posterior longitudinal incision was performed at T3–T4 and L3–L4. The tip of the spinous processes were partially removed. Conventional subdermal EEG needle electrodes, approximately 23 gauge and made of stainless steel, were inserted into the base of spinous processes at the following sites: T3–T4, and at L3–L4. Another needle electrodes, which acted as reference, were placed subdermally at the same level. The sciatic nerve was then exposed through an incision in the hind limb, keeping surrounding muscles intact, and the stimulating electrode placed directly in contact with the nerve. Likewise, two recording electrodes were placed over the gluteus or soleus muscles, in a belly tendon arrangement. A separate ground electrode was placed in the mid-portion of the back (Fig. 1). The electrodes were secured with sutures in the muscle and subcutaneous tissues to prevent movement during surgery. In the supine position a laterocervical incision of the neck was made to allow identification of the cervical vessels An intravenous infusion of isotonic saline solution at a rate of 5 ml/min was administered during the operation to replace surgical losses. Systemic blood pressure was monitored by catheterising the carotid artery. A warming blanket and heating lamp were used to maintain a constant rectal temperature, and the electrocardiogram was monitored continuously. After local infiltration of mepivacain hydrochloride (10 mg/kg) at the abdominal alba line, the infrarenal aorta was approached by a xiphopubic midline laparotomy. After systemic heparinisation (1.5 mg/kg intravenously), the aorta was clamped for 21 min. Evoked potentials were recorded continuously. When the infrarenal aorta was unclamped, evoked potentials were evaluated for at least 30 min postclamping. Finally, the electrodes were removed and the wounds sutured. The animals were placed in cages for neurological evaluation at 24 h, and graded according to the clinical status into two categories: (1) normal: animals with complete recovery (Tarlov's criteria grade IV); (2) paraplegia: animals with no movement of hind limbs or unable to stand or walk (Tarlov's criteria grade 0–III). To determine the correlation between neurologic outcome and evoked potentials we defined the amplitude criterion strictly as absent or present. The rabbits were evaluated by one observer that was blinded to the recording procedure.

Recording procedures

After baseline SEPs, Spinal-EPs, and MEPs were obtained, evoked potentials were recorded continuously

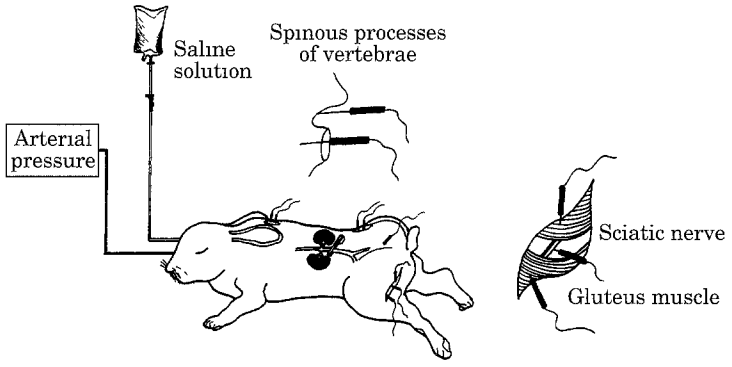

Fig. 1. Schematic drawing showing the experimental model and the sites where the needle electrodes were placed for monitoring of evoked potentials

Table 1. Acquisition parameters for MEPs, Spinal-EPs, and SEPs.

	MEPs	Spinal-EPs	SEPs
Presentation rate	3.2 s	3.2 s	3.2 s
Stimulus duration	0.1 ms	0.1 ms	0.1 ms
Time base	20 ms	20 ms	20 ms
Filter			
Low linear filter	20 Hz	20 Hz	20 Hz
High linear filter	2000 Hz	2000 Hz	2000 Hz
Sensitivity			
Thoracic	50–100 μV	50 μV	50–100 μV
Lumbar	1000–5000 μV	100–200 μV	100–200 μV
Sweeps	1–32	1–32	1–32
Stimulus intensity		9–13 mA	1–3 mA
Twitch	10–15 mA		
High-intensity	45–50 mA (no averaging)		

MEPs motor evoked potentials, Spinal-EPs. spinal evoked potentials, SEPs somatosensory evoked potentials

during and after clamping of the aorta with a clinical evoked potential system (Medelec, Mystro, MS-25, U.K.). SEPs were elicited by stimulating the sciatic nerve and were recorded at L3–L4 and T3–T4. Spinal-EPs were elicited by stimulating the spinal cord at T3–T4 and recording at L3–L4. MEPs were elicited by stimulating the spinal cord at T3–T4 and L3–L4, and recording responses over the muscles gluteus or soleus. Routine parameters for eliciting and recording the SEPs, Spinal-EPs, and MEPs were used (Table 1). Evoked potentials were averaged and displayed on the monitor. According to the intensity of motor stimulus the animals were subdivided into two groups: (1) animals that underwent a minimal intensity of stimulus to evoke a stable motor action potential and twitching of the paraspinal muscles ($n=15$); and (2) animals that underwent a high-intensity of stimulus ($n=15$). In this second group, in which the limbs of the animals were secured to prevent movement artifact, no averaging was necessary

Histological study

The animals were killed at the completion of the project (neurologic evaluation at 24 h) with a lethal intravenous dose of phenobarbital and potassium chloride The spinal cord (proximal and distal to the level of aortic occlusion) was removed for fixation in 10% neutral buffered formalin for at least 10 days. After embedding in paraffin wax, the specimens were sectioned at 5 μm and sections stained with haematoxylin and eosin

Results

Proximal carotid arterial pressure was similar in all groups at baseline (95 ± 15 mmHg) With aortic occlusion, a significant increase ($p<0.05$) was observed in proximal pressures in all groups of rabbits compared with respective baselines (125 ± 10 mmHg). No differences were observed between the group with twitch

stimulus and high intensity stimulation. After removal of the aortic clamp, the proximal blood pressure stabilised to a point not significantly different from the baseline value. The body temperature did not change during the ischaemia time.

Spinal-EPs

Spinal-EPs showed a multiphasic waveform with a negative-positive deflections. Spinal-EPs demonstrated a decrease of amplitude with no shift of latency and no change of waveform to the cord ischaemia. These recordings were never totally lost.

SEPs

SEPs after stimulation of the sciatic nerve were different in morphology depending on the level of recording over the spinal cord. SEPs from lumbar levels usually consisted of bi- or triphasic waves. Over the thoracic spine, SEPs were generally polyphasic consisting of two major negative peaks, and subsequent multiple rippling. The wave amplitudes were more sensitive to ischaemia than their latencies, that is, with the ischaemia the component waves of the SEPs became smaller and their latencies increased. However, only in five animals (17%) the SEPs were completely absent during the ischaemic period. The SEPs component waves returned after declamping of the aorta in reverse order of their disappearance, and were identificable in all animals after reperfusion, regardless of their neurologic outcome. The latency of individual waves normalised faster than the amplitude

MEPs

The compound muscle action potentials after stimulation of the spinal cord generally consisted of simple negative-positive biphasic waves. Increasing the intensity of the stimulus enhanced the amplitude, so the animals with a high level of intensity showed a greater amplitude than the group with twitch stimulus, and required no averaging. Likewise, the amplitude of the MEPs varied considerably, depending on the location of the stimulus electrodes: MEPs elicited by stimulating L3–L4 were higher than stimulating T3–T4. At the onset of ischaemia, the characteristic change of MEPs was a reduction of amplitude, but with no shift of latency. However, MEPs were always abolished during the aortic occlusion, demonstrating that the MEPs recording is especially sensitive to spinal cord ischaemia. The average time elapsed till disappearance was 10.4 ± 1.3 min in the group with twitch stimulus, and 15.6 ± 2.7 min in the group with supramaximum intensity. A clear sensory-motor dissociation was showed by simultaneous recording of evoked potentials during the aortic occlusion (Fig. 2). MEPs were absent in all animals (Fig. 2c, 2d) while SEPs (Fig. 2a) and Spinal-EPs (Fig. 2b) still remained. MEPs recorded at high spine (Fig. 2d) were more sensitive to ischaemia than MEPs at low spine (Fig. 2c), since the high spine response was lost first. After declamping, MEPs by low spine stimulation (Fig. 2c) returned earlier than MEPs by high spine stimulation (Fig. 2d). The average time for the return of waves was dependent upon the stimulus parameters used to record the MEPs: earlier with high intensity stimulation (7.9 ± 1.0 min) than with twitch stimulus (15 ± 1.3 min).

Neurological outcome

At 24 h, 15 animals remained paraplegic (50%). No animals had loss of Spinal-EPs elicited by the stimulation of T3–T4 during and after declamping of the aorta. The sensory component waves of Spinal-EPs were the least sensitive to spinal cord ischaemia, and its presence had no correlation with the final clinical status (15 false negatives, 15 true negatives). Only five animals lost completely the SEPs record during the aortic occlusion, although none of them had neurologic deficit at 24 h. Changes in the amplitudes and latencies of the SEPs did not predict paraplegia. The return of SEPs after reperfusion did not correlate with neurological outcome because all animals showed return of signals immediately after declamping (15 false negatives, 15 true negatives). The overall sensitivity, specificity, and overall accuracy of the loss of Spinal-EPs and SEPs at predicting neurological injury were 0%, 100%, and 0%, respectively.

Table 2 shows the relationship between motor

Fig. 2. (overleaf) Sensory-motor dissociation by simultaneous recording of evoked potentials The motor action potentials were always absent after clamping of the aorta (2c, 2d) while the SEPs (2a) and Spinal-EPs (2b) still remained MEPs at thoracic level (2d) were more sensitive to ischaemia than MEPs at lumbar level (2c), since the high spine response was lost almost immediately after clamping of the aorta After declamping, MEPs by low spine stimulation (2c) returned earlier than MEPs by high spine stimulation (2d) The presence of MEPs was associated with normal neurologic function in this animal with twitch stimulus (case no 3)

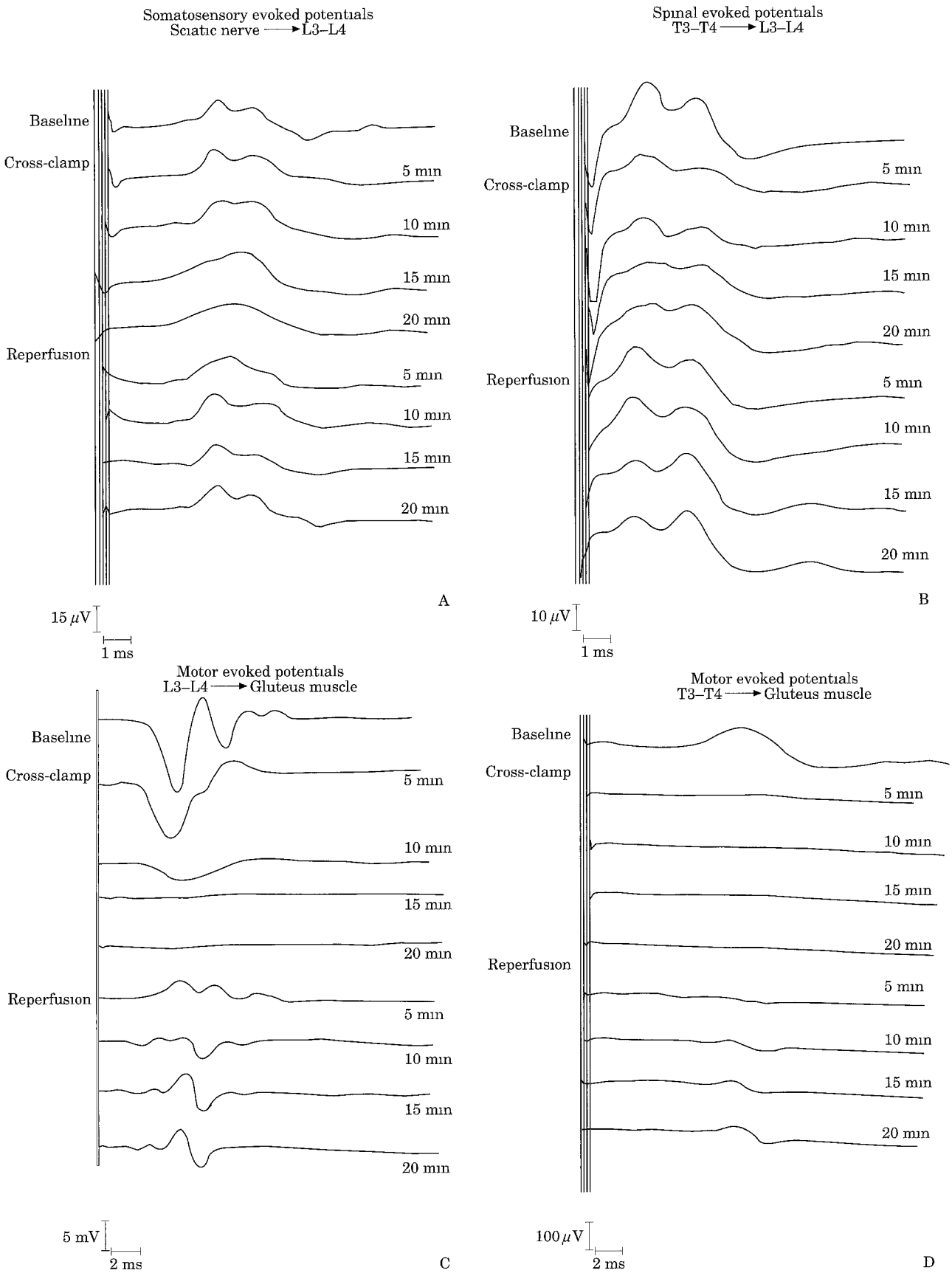
Somatosensory evoked potentials
Sciatic nerve ⟶ L3–L4
Baseline
Cross-clamp
5 min
10 min
15 min
20 min
Reperfusion
5 min
10 min
15 min
20 min
15 μV
1 ms
A
Spinal evoked potentials
T3–T4 ⟶ L3–L4
Baseline
Cross-clamp
5 min
10 min
15 min
20 min
Reperfusion
5 min
10 min
15 min
20 min
10 μV
1 ms
B
Motor evoked potentials
L3–L4 ⟶ Gluteus muscle
Baseline
Cross-clamp
5 min
10 min
15 min
20 min
Reperfusion
5 min
10 min
15 min
20 min
5 mV
2 ms
C
Motor evoked potentials
T3–T4 ⟶ Gluteus muscle
Baseline
Cross-clamp
5 min
10 min
15 min
20 min
Reperfusion
5 min
10 min
15 min
20 min
100 μV
2 ms
D

Table 2. Relationship between motor evoked potentials and neurological outcome.

Clinical status	Response	Sensitivity	Specificity	Predictive value
MEPs ($n=30$)				
a Twitch stimulus ($n=15$)				
a1 Low spine MEPs				
Present paraplegia	0 FN	100%	62.5%	70%
Present normal	5 (33 3%) TN			
Absent paraplegia	7 (46 6%) TP			
Absent normal	3 (20%) FP			
a2 High spine MEPs				
Present paraplegia	0 FN	100%	100%	100%
Present normal	8 (53 3%) TN			
Absent paraplegia	7 (46 6%) TP			
Absent normal	0 FP			
b High-intensity of stimulus ($n=15$)				
b1 Low spine MEPs				
Present paraplegia	7 (46 6%) FN	12 5%	85 7%	50%
Present normal	6 (40%) TN			
Absent paraplegia	1 (6 6%) TP			
Absent normal	1 (6 6%) FP			
b2 High spine MEPs				
Present paraplegia	4 (26 6%) FN	50%	85 7%	80%
Present normal	6 (40%) TN			
Absent paraplegia	4 (26 6%) TP			
Absent normal	1 (6 6%) FP			

MEPs motor evoked potentials, FN false negative, TN true negative, TP true positive, FP false positive

evoked potentials after declamping and neurological outcome. After declamping, low spine MEPs returned in five animals in the group with twitch stimulus (true negatives), and in 13 animals in the group with high intensity stimulation (seven false negatives, and six true negatives). The presence of MEPs by low spine stimulation was associated with normal neurological function in the group with twitch stimulus (0% of false negatives), but this relationship was not consistent in the group with high intensity stimulation (47% of false negatives). High spine MEPs evoked by twitch stimulation was the best predictor of clinical outcome (0% of false negatives, 0% of false positives). However, this relationship was not consistent with high intensity stimulation (27% of false negatives, 7% of false positives). The average time until loss of MEPs and time until return during reperfusion did not predict neurological injury. The overall sensitivity, specificity, and overall accuracy of the loss of MEPs at predicting paraplegia were, respectively. 100%, 63%, and 70% in the group with twitch stimulus at low spine; 100%, 100%, and 100% in the group with twitch stimulus at high spine; 13%, 86%, and 50% in the group with high intensity stimulation at low spine; and 50%, 86%, and 80% in the group with high intensity stimulation at high spine

Histopathology

The histopathological findings clearly correlated with the neurologic outcome. Paraplegic animals showed pronounced vacuolisation and gliosis almost entirely confined to the anterior horn, while the dorsal columns were generally well preserved (Fig. 3). This pattern of diffuse destruction of gray matter was observed in all experiments, although the total number of ischaemia reperfusion-damaged neurons manifested by somatic and dendritic argyrophilia was not quantified. The spinal cord structure was intact in animals that were spared neurologic deficit. No infarctions were detected in this group. Normal animals showed ganglion cells in anterior horns with normal nuclei and cytoplasm, which were indistinguishable from those in non-ischaemic zones of the cord.

Discussion

Estimating spinal cord ischaemia during surgery and predicting possible paraplegia poses a difficult task for surgeons. It is impossible to predict what aortic clamping time can be tolerated in a particular patient

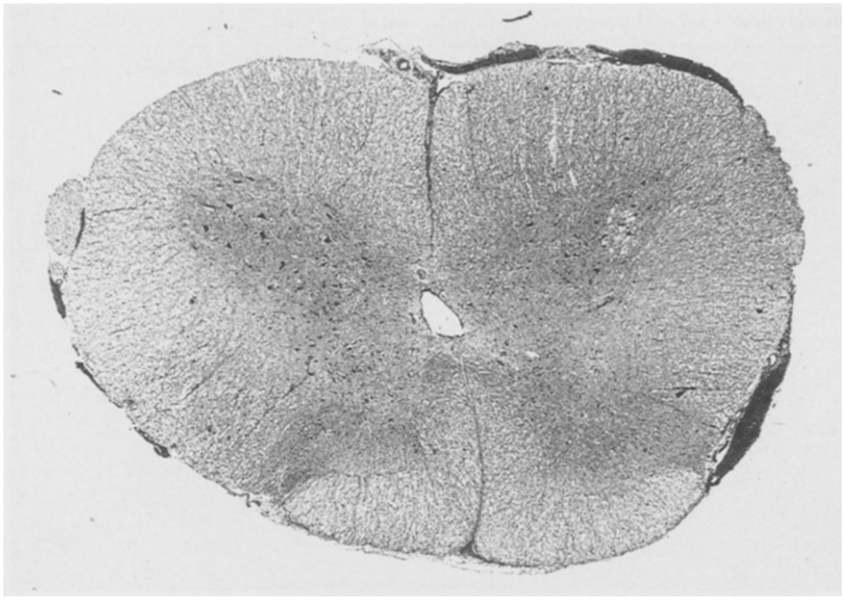

Fig. 3. Photomicrograph of lumbar spinal cord of a paraplegic animal Histologically there is pronounced vacuolisation and cell degeneration confined to the anterior horn, while the dorsal horns are well preserved (haematoxylin-eosin stain, original magnification ×10)

because the exact vascular anatomy, including collateral vessels that provide the spinal cord with blood is unknown. Neurophysiologic monitoring has been employed to alert the surgeon of impaired spinal cord function and the risk of permanent spinal cord injury, but currently available methods only monitor the sensory pathways. Thus, it is not surprising to find case reports of postoperative motor deficits despite normal evoked potentials throughout the operation. These false-negative results may occur because a lesion may reside outside of the tract monitored by the test or because the test was not sensitive enough to detect the impaired spinal cord function.[16,17] This study provides important information on the efficacy of the motor-evoked potentials as a monitor of spinal cord function in a rabbit spinal cord ischaemia model of infrarenal aortic occlusion. MEPs were always abolished, demonstrating that the MEPs recording are specially sensitive to spinal cord ischaemia. In addition, the return of MEPs at thoracic level was predictive of normal spinal cord function.

Motor evoked potential monitoring usually involves stimulation either of the cerebral cortex or directly from the spine, with recordings made from the spinal cord, peripheral nerve or muscle. In this study, we monitored MEPs with recording electrodes placed in muscles of the hind limb and direct stimulation of the spinal cord through needle electrodes inserted into the bone of the spinous processes of vertebrae after their tip had been cut off. This technique is a modification of a previous method reported by Owen *et al.*,[23] who described a neurogenic motor evoked potential from electrodes placed over sciatic nerve after stimulating the spinal cord via spinous processes. This procedure is significantly less invasive that direct stimulation of the spinal cord using epidural electrodes, and may be clinically applicable for intraoperative monitoring of spinal cord motor tract function.[19]

We observed that the motor action potential was always absent after clamping of the aorta while the Spinal-EPs and SEPs still remained. This confirms the dissociation between the motor and sensory pathways, and that the anterior horn of the spinal cord is more sensitive to ischaemia than the dorsal horn. Therefore, the anterior horn may become ischaemic during aortic occlusion, while the sensory evoked potentials are still normal, which explains the false-negative results reported in the literature. This is consistent with the histopathological examination of paraplegic animals that showed pronounced vacuolisation and gliosis almost entirely confined to the anterior horn, while the dorsal columns were generally well preserved. Although the component waves of the SEPs became smaller and their latencies increased, they only disappeared with severe ischaemia. Thus, SEPs may not detect less severe ischaemia that is limited to the motor system. After declamping of the aorta, the SEPs waveform returned in reverse order of their disappearance, and were identifiable in all animals, regardless of the neurologic outcome. Because of the differences in anatomical origin and sensitivity, the SEPs do not appear to be a good predictor of clinical motor status. On the other hand, Spinal-EPs were the

least sensitive to spinal cord ischaemia, because they were never totally lost, and the waveforms only showed a reduction of amplitude with no shift of latency. Although these results with sensory evoked potentials (false-negative rate of 50%) are in agreement with the experimental data reported by other researchers,[24,25] they are in disagreement with some clinical reports.[10] One explanation for the inconsistency between experimental and clinical reports might be the neuroanatomic differences between the models, and the techniques employed. Because of this, we should be cautious when extrapolating our results.

The intensity of stimulus appears to be the most important factor in relation to the signal recorded and the overall accuracy of the loss of MEPs at predicting neurologic injury. MEPs at high spine were lost almost immediately after clamping of the aorta, while MEPs at low spine remained, suggesting that the evoked potential at thoracic level are the most sensitive to ischaemia, since the time required to disappear was shorter. Although the presence or absence of MEPs at thoracic level with twitch stimulus was always correlated with the clinical outcome, this relationship was not consistent in the animals with high-intensity stimulation (27% of false negatives, and 7% of false positives). Therefore, in our experimental model, high spine MEPs evoked by twitch stimulus was the most useful method for real-time evaluation of spinal cord ischaemia, and the best predictor of clinical outcome during reperfusion, because all animals that were not paraplegic showed thoracic responses (0% of false negatives), and its absence was associated with neurologic deficit (0% of false positives).

It remains to be seen if our findings are directly applicable to operation for thoracoabdominal aneurysms in humans. It is likely that monitoring of the motor action potential will enhance the detection of the spinal cord ischaemia, and will be useful to prevent neurologic dysfunction, at the time when it is still reversible by reimplantation of intercostal vessels. Stimulation of the spinal cord proximal to the clamping site and recording a distal muscle evoked potential, is relatively non-invasive, and it is easy to perform in humans. To activate descending motor pathways, spinal cord stimulation can be used as an alternative method to cortical stimulation, because the transcranial electrical stimulation is greatly influenced by the majority of anaesthetic agents and the dosages employed, exerting their depressing effect more at cortical than at spinal level.[26] By stimulating at thoracic level with the minimum intensity necessary to evoke a stable motor potential, and keeping it constant during surgery, the sensitivity and specificity of MEPs should be able to accurately monitor spinal cord ischaemia during thoracoabdominal aneurysm repair.

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